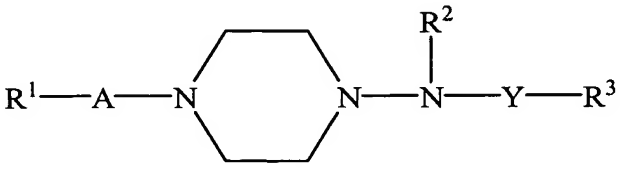
IN THE CLAIMS

Please amend the claims as follows:

Claim 1 (Withdrawn): A method for specifically potentiating an N-type Ca^{2+} channel activity, which method comprises administering an effective amount of a compound of the following formula (I):



(I)

wherein R^1 is lower alkyl, aryl, ar(lower)alkoxy or a heterocyclic group, the above groups being optionally substituted by halogen, R^2 is hydrogen atom or lower alkyl, R^3 is cyclo(lower)alkyl, aryl or ar(lower)alkyl, the above groups being optionally substituted by halogen, A is -CO-, $-SO_2-$ or lower alkylene, and Y shows -CO-, $-SO_2-$ or -CONH-, a salt thereof, a prodrug thereof or a solvate thereof to a subject.

Claim 2 (Withdrawn): The method of Claim 1, wherein the compound of the formula (I) is N-(4-acetyl-l-piperazinyl)-p-fluorobenzamide monohydrate.

Claims 3-8 (Cancelled)

Claim 9 (Withdrawn): A method for screening a compound having an effect of specifically potentiating an N-type Ca^{2+} channel activity comprising:

bringing a neuronal voltage-dependent calcium channel α_{1B} subunit expression cell into contact with a test compound;

measuring a membrane current of the cell;

bringing a neuronal voltage-dependent calcium channel α_{1B} non-expression cell into contact with a test compound;

measuring a membrane current of the non-expression cell; and

comparing the membrane current of the aforementioned expression cell and the membrane current of the non-expression cell.

Claim 10 (Withdrawn): The method of Claim 9, wherein the neuronal voltage-dependent calcium channel α_{1B} non-expression cell is a cell made to express a neuronal voltage-dependent calcium channel α_{1A} or α_{1E}.

Claim 11 (Withdrawn): The method of Claim 9, wherein the expression cell is *Xenopus* oocyte made to express a neuronal voltage-dependent calcium channel α_{1B} subunit.

Claim 12 (Withdrawn): The method of Claim 10, wherein the expression cell is *Xenopus* oocyte made to express a neuronal voltage-dependent calcium channel α_{1B} subunit.

Claim 13 (Withdrawn): The method of Claim 9, wherein the neuronal voltage-dependent calcium channel α_{1B} non-expression cell is *Xenopus* oocyte made to express a neuronal voltage-dependent calcium channel α_{1A} or α_{1E}.

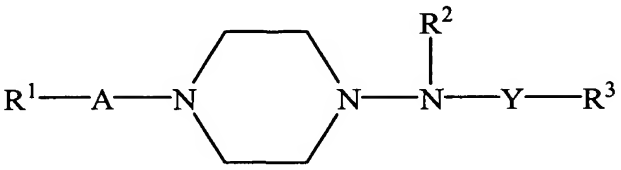
Claim 14 (Withdrawn): The method of Claim 11, wherein the neuronal voltage-dependent calcium channel α_{1B} non-expression cell is *Xenopus* oocyte made to express a neuronal voltage-dependent calcium channel α_{1A} or α_{1E}.

Claims 15-20 (Cancelled)

Claim 21 (Previously Presented): A method for treating a brain disorder comprising:

administering to a subject in need thereof an effective amount of a compound having an effect of specifically potentiating an N-type Ca^{2+} channel activity,

wherein said compound is not the compound of formula (I):



(I)

wherein

R^1 is lower alkyl, aryl, ar(lower)alkoxy or a heterocyclic group, the above groups being optionally substituted by halogen,

R^2 is hydrogen atom or lower alkyl,

R^3 is cyclo(lower)alkyl, aryl or ar(lower)alkyl, the above groups being optionally substituted by halogen,

A is -CO-, $-SO_2-$ or lower alkylene, and

Y is -CO-, $-SO_2-$ or -CONH-,

wherein said subject has at least one brain disorder selected from the group consisting of dementia, amnesia, schizophrenia, manic-depressive psychosis, stroke, head trauma, nicotine withdrawal symptom, spinal trauma, anxiety, thamuria, incontinence of urine, myotonic dystrophy, attention deficit hyperactivity disorder, narcolepsy, Parkinson's disease, autism and psychosomatic disorder.

Claim 22 (Previously Presented): The method of Claim 21, wherein said subject has at least one brain disorder selected from the group consisting of senile dementia, Alzheimer's dementia, cerebrovascular dementia, dementia after cerebral trauma, dementia caused by

cerebral tumor, dementia caused by chronic subdural hematoma, dementia caused by normal pressure hydrocephalus, dementia caused by meningitis, and Parkinsonian dementia.

Claim 23 (Previously Presented): The method of Claim 21, wherein said compound is administered in an amount ranging from 0.10 to 10 mg/kg body weight.

Claim 24 (Previously Presented): The method of Claim 21, wherein said subject is a non-human mammal.

Claim 25 (Previously Presented): The method of Claim 21, wherein said subject is human.

Claim 26 (Previously Presented): The method of Claim 21, wherein said compound is administered intravenously.

Claim 27 (Previously Presented): The method of Claim 21, wherein said compound is administered intramuscularly.

Claim 28 (Previously Presented): The method of Claim 21, wherein said compound is administered orally.

Claim 29 (Previously Presented): The method of Claim 21, wherein said compound is administered as a prodrug.

Claim 30 (Previously Presented): The method of Claim 21, wherein said compound has been obtained by a method for identifying a compound which has an effect of specifically potentiating an N-type Ca^{2+} channel activity, comprising:

bringing a neuronal voltage-dependent calcium channel α_{1B} subunit expression cell into contact with a test compound;

measuring a membrane current of the cell;

bringing a neuronal voltage-dependent calcium channel α_{1B} non-expression cell into contact with a test compound;

measuring a membrane current of the non-expression cell;

comparing the membrane current of the aforementioned expression cell and the membrane current of the non-expression cell and

selecting a compound which has an effect of specifically potentiating an N-type Ca^{2+} channel activity.

Claim 31 (Previously Presented): The method of Claim 30, wherein in said method for identifying a compound which has an effect of specifically potentiating an N-type Ca^{2+} channel activity, the neuronal voltage-dependent calcium channel α_{1B} non-expression cell is a cell made to express a neuronal voltage-dependent calcium channel α_{1A} or α_{1E}.

Claim 32 (Previously Presented): The method of Claim 30, wherein in said method for identifying a compound which has an effect of specifically potentiating an N-type Ca^{2+} channel activity, the expression cell is *Xenopus* oocyte made to express a neuronal voltage-dependent calcium channel α_{1B} subunit.

Claim 33 (Previously Presented): The method of Claim 30, wherein in said method for identifying a compound which has an effect of specifically potentiating an N-type Ca^{2+} channel activity, the expression cell is *Xenopus* oocyte made to express a neuronal voltage-dependent calcium channel α_{1B} subunit.

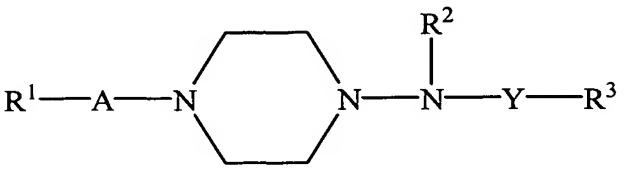
Claim 34 (Previously Presented): The method of Claim 30, wherein in said method for identifying a compound which has an effect of specifically potentiating an N-type Ca^{2+} channel activity, the neuronal voltage-dependent calcium channel α_{1B} non-expression cell is *Xenopus* oocyte made to express a neuronal voltage-dependent calcium channel α_{1A} or α_{1E}.

Claim 35 (Previously Presented): The method of Claim 30, wherein in said method for identifying a compound which has an effect of specifically potentiating an N-type Ca^{2+} channel activity, the neuronal voltage-dependent calcium channel α_{1B} non-expression cell is *Xenopus* oocyte made to express a neuronal voltage-dependent calcium channel α_{1A} or α_{1E}.

Claim 36 (Previously Presented): A method for alleviating a brain disorder comprising:

administering to a subject in need thereof an effective amount of a compound having an effect of specifically potentiating an N-type Ca^{2+} channel activity,

wherein said compound is not the compound of formula (I):



(I)

wherein

R^1 is lower alkyl, aryl, ar(lower)alkoxy or a heterocyclic group, the above groups being optionally substituted by halogen,

R^2 is hydrogen atom or lower alkyl,

R^3 is cyclo(lower)alkyl, aryl or ar(lower)alkyl, the above groups being optionally substituted by halogen,

A is -CO-, $-SO_2-$ or lower alkylene, and

Y is -CO-, $-SO_2-$ or -CONH-.